pressure does not depend therefore upon mechanical factors alone as in acute experiments when the aorta is occluded (Barcroft[9]). Nor can the hypertension in coarctation of the aorta be explained by local mechanical factors such as the increase in resistance offered by the narrowed aorta and the collateral paths around it.[10] It depends upon a reaction of the whole peripheral arteriolar system. Hypertension the result of clamping the aorta is in this respect similar to that following constriction of the renal vessels. In both cases the deciding factor is interference with the dynamics of the renal circulation.

One further remark seems pertinent. The observation of similar consequences to the arterial pressures of constriction of the aorta in man (coarctation of the aorta) and in dogs (clamping of the aorta) warrants the inference that interference with the hemodynamics of the renal blood supply in man may lead to arterial hypertension as Goldblatt has demonstrated that it does in dogs and monkeys.

Summary. Clamping the aorta above the orifices of the renal artery in dogs is followed by elevation of the diastolic level of arterial pressure in the hind legs as well as in the carotid arteries. Constriction of the peripheral arterioles is, therefore, a general phenomenon, just as when it follows partial clamping of the renal arteries. The hypertension which develops in coarctation of the aorta in man is on this evidence analogous to that which accompanies constriction of the renal arteries. The evidence suggests strongly that interference with the hemodynamics of the renal circulation leads to hypertension in man as well as in animals.

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Form of Ventricular Contraction in Cardiac Infarction; Fluoroscopic Studies.

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Infarction of the heart following coronary artery occlusion is attended by a profound disturbance in the circulation. While the circulatory dynamics and electrocardiographic changes have been

[9] Barcroft, H., *J. Physiol.*, 1931, **71**, 281.

[10] Blumgart, H. L., Lawrence, J. S., and Ernstene, A. C., *Arch. Int. Med.*, 1931, **47**, 806.

extensively investigated, very little attention has been paid to direct observation of alterations in cardiac contraction.

That experimental ligation of a coronary artery interferes with the orderly contraction of the ventricles has been known since the 17th century.[1, 2] A detailed description of the changes in ventricular contraction, however, has been lacking until the recent investigation of Tennant and Wiggers.[3] Employing the myocardiograph, these authors found that immediately after occluding branches of the coronary arteries in dogs the area of muscle rendered ischemic ceased to contract and paradoxical (reverse) movements occurred, *i. e.*, the ischemic area bulged passively while the remainder of the ventricle contracted normally.

There have been few recorded fluoroscopic observations in man of the effect of coronary artery occlusion on the contractile movements of the heart, even in cases where the infarct is so extensive as to result in the formation of a ventricular aneurysm.[4, 5, 6] Zadek,[7] and Levene[8] observed localized impairment of contraction of the left ventricle in coronary artery disease but did not describe systolic expansion (reversal of pulsation) which was a characteristic finding in the studies of Tennant and Wiggers on dogs.

The movements of the left ventricle in 64 cases of myocardial infarction due to coronary artery occlusion have been studied by

TABLE I.
Types of Ventricular Contraction in Myocardial Infarction.

	Fluoroscopy	Roentgenkymography
1. Normal pulsation	17	13
2. Reversal of pulsation		
a. complete reversal	26	24
b. partial reversal		
lag of systolic contraction	4	4
doubling of systolic contraction	2	5
3. Impairment of contraction		
a. absence of pulsation	2	5
b. diminution of pulsation	13	13
Total	64	64

1 Chirac, P., *De Motu Cordis, Adversaria Analytica,* 1698, p. 121, cited by Sée, Bochefontaine and Roussy, *Compt. Rend. Acad. d. Sc.,* 1881, **92**, 86.

2 Samuelson, B., *Z. f. Klin. Med.,* 1881, **2**, 12.

3 Tennant, R., and Wiggers, C. J., *Am. J. Physiol.,* 1935, **112**, 351.

4 Sezary, A., and Alibert, J., *Bull. et mem. Soc. med. d. hop. de Paris,* 1922, **46**, 172.

5 Lenk, R., *Fortschr. a. d. Geb. d. Röntgenstrahlen,* 1926, **35**, 1265.

6 Kalisch, Z., *Wien. Klin. Wchnschr.,* 1927, **40**, 1078.

7 Zadek, E., *Klin. Wchnschr.,* 1932, **11**, 1255.

8 Levene, G., Lowman, R. M., and Wissing, E. G., *Am. Heart J.,* 1938, **16**, 133.

TABLE II.
Regions of Left Ventricle Exhibiting Abnormal Contraction.

	Fluoroscopy	Roentgenkymography
Normal	17	13
Upper section left ventricular contour	3	2
Middle " " " "	6	3
Supraäpical " " " "	8	13
Apical " " " "	11	12
Lower half " " "	16	18
Entire " " "	3	3
Total	64	64

fluoroscopy at intervals varying from one month to 4 years after the acute attack. The findings were compared with roentgenkymograms which were taken immediately after fluoroscopy. In addition cine-roentgenographic studies were carried out in a group of 24 subjects with normal hearts and with cardiac infarcts.

The fluoroscopic observation was performed with the patient in the postero-anterior position, visualizing the anterior wall and apex

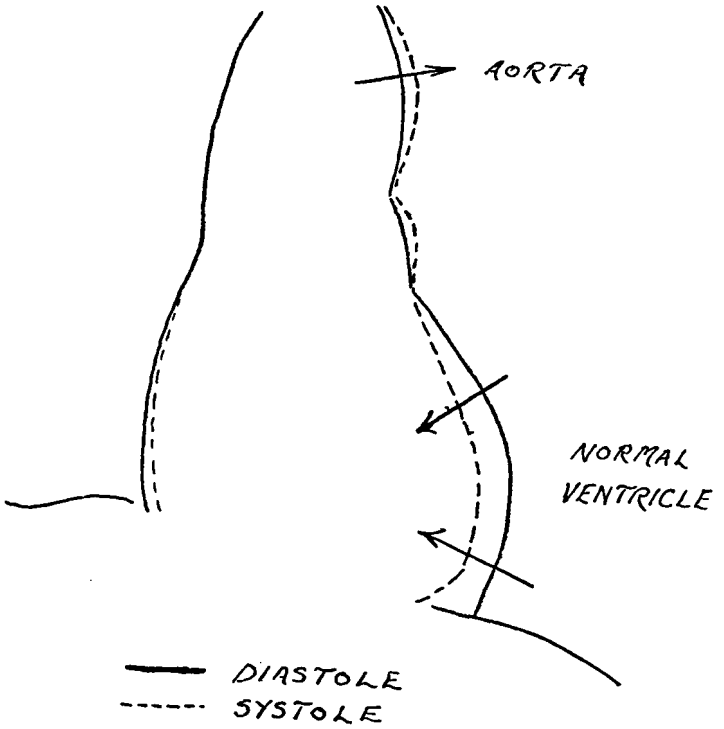

FIG. 1A.

Normal Contraction. Entire left ventricle contracts during systole. There is an inthrust of the ventricle synchronous with expansion of the aorta.

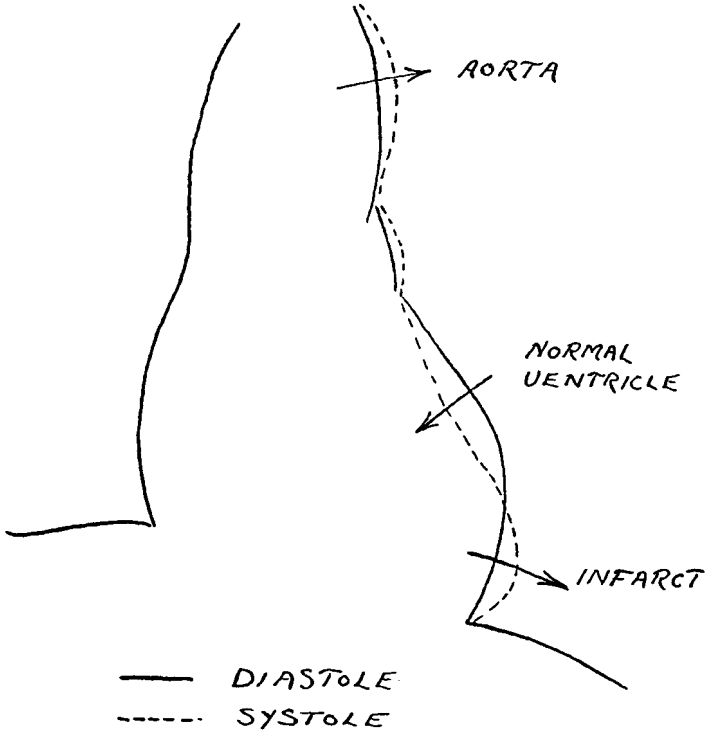

FIG. 1B.

Reversal of Contraction. While the upper normal portion of the left ventricle contracts (inthrust), the diseased lower apical muscle is passively expanded (outthrust).

of the left ventricle. Time relationships of the pulsations were established by comparing the movements of the left ventricle with those of the aorta and pulmonary artery. The arterial pulsations are normally in opposite phase to those of the ventricle, the aorta expanding in systole while the ventricle contracts (Fig. 1A). Another method for establishing time relationships was auscultation of the heart by means of a Bowles diaphragm held to the patient's chest at the left costal margin in the fourth interspace. Normally there is observed an inthrust of the entire left ventricular contour and elevation of the apex and diaphragmatic surface of the heart synchronous with the expansion of the aorta and the first heart sound (Fig. 1A).

The contractile movements of the heart were studied at the end of a deep inspiration. In addition to immobilizing the diaphragm this procedure also slows the heart rate. This is of advantage since rapid heart action makes visualization of the details of ventricular move-

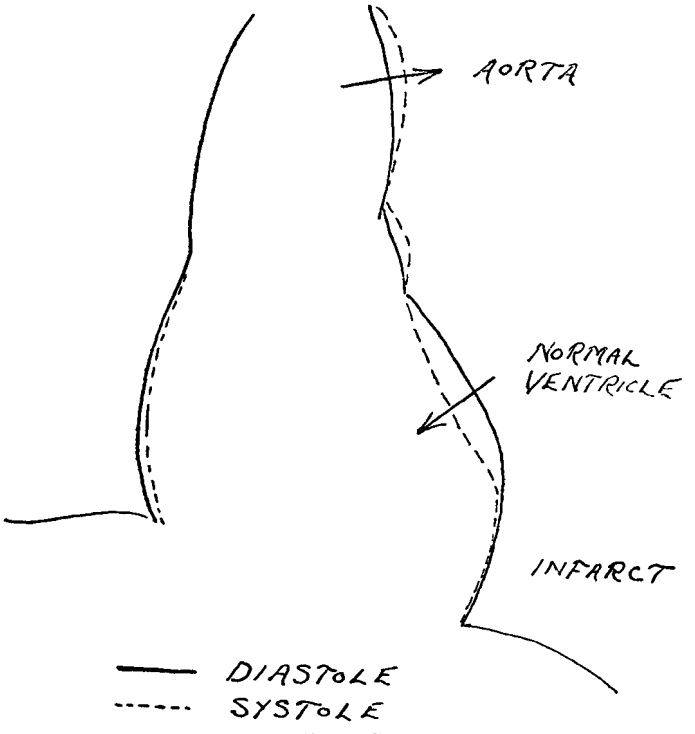

FIG. 1C.

Absence of Pulsation. While the normal upper portion contracts (inthrust), no movement is seen in the affected lower half.

ment difficult. It was also found helpful to magnify the movements by drawing the screen away from the patient, particularly when they were of small amplitude. The presence of apical pericardial fat may obscure muscle movement and give the impression of diminution or absence of pulsation. One must therefore look within the fat pad to observe muscle contraction.

Abnormalities in contraction were observed on fluoroscopy in 47 of the 64 cases studied (73%). The types of movement observed are classified in Table I. The fluoroscopic observations corresponded closely with roentgenkymographic findings both in regard to the type of movement (Table I) and the region of the left ventricle involved (Table II). The lower half of the left ventricle, particularly the apical and supraäpical segments, most frequently exhibited these abnormalities in contraction.

Reversal of pulsation was the most common and definite abnormality of ventricular contraction associated with myocardial infarction (50%). It frequently appeared as a wavelike movement

along the border of the left ventricle. As the intraventricular tension abruptly rose in the isometric phase of systole the weakened myocardium at the site of the infarct passively expanded (outthrust), while the normal region of the ventricle contracted (inthrust) (Fig. 1B). This phenomenon was also observed in 3 cases of ventricular aneurysm. Occasionally the reversal of pulsation was not complete but appeared as a definite lag of systolic inthrust or as a double systolic pulsation.

Localized impairment of contraction was observed in 23% of the cases. This appeared as a marked diminution or absence of pulsation (Fig. 1C). These changes may occur in a region where the infarcted myocardium is not sufficiently powerful to contract vigorously but is able to withstand the intraventricular pressure.

These localized abnormalities in pulsation have not been observed by us in normal individuals or in those with other types of heart disease than coronary artery disease. The pulsations are occasionally much diminished, or even absent, in the apical region when the heart is markedly enlarged, but such cases were not included in the present series.

In this report we have presented the abnormalities in contraction of the left ventricle as observed in the postero-anterior view alone. In addition, the movements of the postero-lateral wall of the left ventricle in myocardial infarction are being studied in the left oblique and left lateral positions.

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Basal Heat Loss and Production in Women at Temperatures From 23°C to 36°C.

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Recent work[1, 2, 3] on the effect of environmental temperature on heat loss in humans has been restricted to male subjects, most of them

[1] Hardy, J. D., and Du Bois, E. F., *J. Nutrition*, 1938, **15**, 461.

[2] Winslow, C.-E. A., Herrington, L. P., and Gagge, A. P., *Am. J. Physiol.*, 1937, **120**, 1.

[3] Hardy, J. D., and Soderstrom, G. F., *J. Nutrition*, 1938, **16**, 5, 493.